

Emotion Context Insensitivity in Major Depressive Disorder

Jonathan Rottenberg
University of South Florida

James J. Gross and Ian H. Gotlib
Stanford University

The present study tested 3 competing views of how depression alters emotional reactivity: positive attenuation (reduced positive), negative potentiation (increased negative), and emotion context insensitivity (ECI; reduced positive and negative). Normative and idiographic stimuli that elicited happy, sad, and neutral states were presented to currently depressed, formerly depressed, and healthy control individuals while experiential, behavioral, and autonomic responses were measured. Currently depressed individuals reported less sadness reactivity and less happiness experience across all conditions than did the other participants, and they exhibited a more dysphoric response to idiographic than to normative stimuli. Overall, data provide partial support for the positive attenuation and ECI views. Depression may produce mood-state-dependent changes in emotional reactivity that are most pronounced in emotion experience reports.

Keywords: depression, emotional functioning, reactivity

Major depressive disorder (MDD) is a devastating, sometimes fatal, psychiatric condition that afflicts nearly one fifth of the population (Kessler, 2002). The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* classifies MDD as a mood disorder (American Psychiatric Association [APA], 1994). Indeed, durable disturbance of mood is one of the most salient features of MDD. *DSM-IV* diagnostic criteria specify symptoms of at least 2 weeks' duration that implicate deficient positive affect (e.g., anhedonia), excessive negative affect (e.g., guilt), or both. Consistent with these diagnostic criteria, depressed patients consistently report low levels of positive affect and high levels of negative affect on questionnaire and interview measures (Clark, Watson, & Mineka, 1994).

Researchers studying depression have increasingly sought to clarify how this mood disorder alters emotional reactivity. One problem in addressing this question is that the core terms of *mood* and *emotion* are notoriously slippery and have often been used in confusing and contradictory ways. Following contemporary affective science (e.g., Rottenberg & Gross, 2003), here we refer to *moods* as diffuse, slow-moving feeling states that are weakly tied to specific objects or elicitors (Watson, 2000). In contrast, *emotions* refer to quick-moving reactions that occur when an organism processes a meaningful stimulus that exercises its adaptive capacities (e.g., Ekman, 1992; Tooby & Cosmides, 1990). Emotional reactions typically involve coordinated changes in emotional feel-

ings, behavior, and physiology and last for seconds or minutes (Keltner & Gross, 1999). Again by contrast, moods last for hours or days and exert their clearest effects on feelings and cognitions (as opposed to behavior or physiology). Using these definitions, we see that the syndrome of depression necessarily involves changes in mood but does not require changes in emotional reactions.

Most conceptions of the relation between moods and emotions view these constructs as linked, with moods altering the probability of having specific emotions. More specifically, moods are generally thought to potentiate like-valenced or matching emotions (e.g., irritable mood facilitates angry reactions; Rosenberg, 1998). If this logic were to be extended to clinical mood states, one would expect depressed persons' lack of positive mood to attenuate positive emotional reactivity and their high levels of negative mood to potentiate negative emotional reactivity. In the following sections, we review experimental evidence that bears on these predictions.

Positive Attenuation Hypothesis

Depressed persons' low positive mood is the starting point for the positive attenuation hypothesis—that depressed persons will exhibit attenuated emotional reactivity to positive emotional stimuli. Indeed, one of the cardinal symptoms of this disorder is anhedonia (the reduced ability to experience pleasure). Moreover, depressed individuals exhibit several other symptoms that are also indicative of deficient appetitive motivation (e.g., psychomotor retardation, fatigue, anorexia, apathy) and that are interpretable in terms of a reduced responsiveness to appetitive stimuli and/or a reduced drive to engage with positive or rewarding features of the environment. In fact, several theorists center their accounts of emotion dysregulation in MDD around this constellation of appetitive deficits (e.g., Clark & Watson, 1991; Clark et al., 1994; Depue & Iacono, 1989; Henriques & Davidson, 1991).

Investigators have obtained relatively robust support for the positive attenuation hypothesis. For example, compared with non-

Jonathan Rottenberg, Department of Psychology, University of South Florida; James J. Gross and Ian H. Gotlib, Department of Psychology, Stanford University.

This research was supported by National Institute of Mental Health grants awarded to Ian H. Gotlib (MH59259) and James J. Gross (MH58147). We express our appreciation to Adrine Biuckians, Jennifer Champion, and Kathryn Dingman for their help in conducting this study.

Correspondence concerning this article should be addressed to Jonathan Rottenberg, Department of Psychology, University of South Florida, PCD 4118G, 4202 East Fowler Avenue, Tampa, FL 33620-7200. E-mail: jrottenb@cas.usf.edu

depressed control participants, depressed individuals have been shown to be characterized by attenuated reactivity with respect to experience reports when responding to slides depicting pleasant scenes (e.g., Allen, Trinder, & Brennen, 1999; Sloan, Strauss, Quirk, & Sajatovic, 1997; Sloan, Strauss, & Wisner, 2001) and to an amusing film clip (Rottenberg, Kasch, Gross, & Gotlib, 2002). Depressed individuals have been found to exhibit less positive emotion—expressive behavior in response to pleasant film and drink stimuli (Berenbaum & Oltmanns, 1992) and pleasant slides (Sloan et al., 2001) and to be less behaviorally responsive to reward contingencies (Henriques & Davidson, 2000). In addition, recent evidence suggests that positive words evoke less neural reactivity relative to neutral words in depressed individuals than is the case in nondepressed control participants (as measured by functional MRI; Canli et al., 2004). In sum, a growing body of work suggests that deficits in response to positive, approach-related emotion cues are a distinguishing characteristic of depressed individuals.

Negative Potentiation Hypothesis

Likewise, depressed persons' high negative mood is the starting point for the negative potentiation hypothesis—that depressed persons will exhibit potentiated emotional reactivity to negative emotional stimuli. Clinicians have long commented on depressed persons' pervasive negativity of thought and affect, evidenced in negative beliefs about the self, world, and future (Beck, 1967), and observations of outward expressions of negative emotion, such as crying (APA, 1994). Perhaps most explicitly, cognitive theorists have advanced a view of depression in which negative moods and negative emotions are mutually reinforcing (e.g., Beck, 1967; Beck, Rush, Shaw, & Emery, 1979). Beck's schema model and related theories of depression (e.g., Bower, 1981) conceptualize the disorder in terms of cognitive structures, or schemas, that serve to negatively distort the processing of emotional stimuli. It is important to note that, according to these theories, negative mood states prime, or activate, these cognitive structures. Once activated, these structures precipitate depressotypic emotional responses (e.g., acute feelings of sadness) whenever schema-matching negative emotion stimuli are encountered, presumably potentiating reactivity to negative emotional stimuli in MDD.

It is somewhat surprising that empirical support for the negative potentiation hypothesis is limited. One study found clinically depressed individuals to exhibit greater electrodermal reactivity to negative social scenarios than did healthy control participants (Sigmon & Nelson-Gray, 1992). Perhaps the strongest support for the negative potentiation hypothesis has been obtained from "analog" studies of dysphoric persons (Golin, Hartman, Klatt, Munz, & Wolfgang, 1977; Lewinsohn, Lobitz, & Wilson, 1973), but this support may not generalize to diagnosed depression (e.g., Gotlib, 1984). Finally, other data appear to flatly contradict the negative potentiation hypothesis. For example, depressed persons appear to exhibit diminished amygdala response to fearful faces (Thomas et al., 2001), diminished electrodermal and startle reactivity to loud noise (e.g., Allen et al., 1999; Lader & Wing, 1969), and a diminished pain report to a range of noiceptive stimuli (reviewed in Dickens, McGowan, & Dale, 2003).

Emotion Context-Insensitivity Hypothesis

If we take seriously both the data that support positive attenuation and the data that contradict negative potentiation, what are the implications for researchers' overall picture of emotional reactivity in MDD? One possibility is that a third perspective is needed to integrate existing data, a view that posits that depressed persons exhibit diminished emotional reactivity to positive stimuli (agreeing with the positive attenuation hypothesis) and diminished emotional reactivity to negative stimuli (disagreeing with the negative potentiation hypothesis). We refer to this hypothesis of valence-independent deficits in emotional reactivity in MDD as the *emotion context-insensitivity* (ECI) hypothesis.

The ECI hypothesis (Rottenberg & Gotlib, 2004) is derived from evolutionary accounts of depression that view this syndrome as strongly characterized by disengagement (Klinger, 1975; Nesse, 2000). In particular, the ECI hypothesis assumes that depressed mood states powerfully influence ongoing responses to the environment. For example, according to Nesse (2000), depressed mood states evolved as an internal signal designed to bias an organism against action. That is, depressed mood evolved originally as a defensive response to adverse situations in which continued activity might prove to be futile or dangerous (e.g., famine). Pessimism, self-absorption, and loss of interest in the environment are all features of depression that hold a person in place and prevent ill-considered actions. In other words, depressed mood states are postulated to prompt withdrawal and broad reductions in motivated activity, which encompass reduced reactivity to novel positive or negative emotional stimuli.

Though a novel hypothesis, ECI makes contact with several naturalistic observations of depressed individuals. For example, ECI is consistent with the phenomenology of depression, which often features perceptions of the world as flat, dull, and empty and the conviction that "everything is the same" (Healy, 1993); ECI is also consistent with ward observations of depressed inpatients in which depressed persons have been described as being characterized by few changes in expressive behavior to a range of environmental events (see also Rottenberg & Gotlib, 2004).

Perhaps the strongest empirical evidence consistent with ECI has emerged from experiments using within-subjects designs in which study participants are exposed to both positive and negative emotional stimuli. For example, compared with nondepressed control participants, depressed persons have been found to exhibit less affective modulation of startle during affective picture viewing (Allen et al., 1999; Dichter, Tomarken, Shelton, & Sutton, 2004), less electromyography (EMG) modulation during affective imagery (Schwartz, Fair, Salt, Mandel, & Klerman, 1976; Gehricke & Shapiro, 2000; Greden, Genero, Price, Feinberg, & Levine, 1986), less EMG modulation in response to expressive facial stimuli (Wexler, Levenson, Warrenburg, & Price, 1994), less valence-related modulation of event-related brain potentials (Deldin, Keller, Gergen, & Miller, 2001); less differential neural responding to valenced emotion face stimuli (Gotlib, Sivers, Canli, Kasch, & Gabrieli, 2001), less reported sadness reactivity and lower levels of amusement to sad and amusing films (Rottenberg, Kasch, et al., 2002), and blunted autonomic responding to a variety of stimuli (e.g., Dawson, Schell, & Catania, 1977).

Several emotion theorists have argued that the capacity to react to changing stimuli with appropriate emotions is critical for guid-

ing successful adjustment to the environment (Keltner & Gross, 1999; Lazarus, 1991). In this sense, one would expect the diminished emotional reactivity in ECI to be associated with poor adaptation. Although there has been little investigation of the clinical significance of ECI, early evidence suggests that insensitivity to changing environment contexts in MDD may herald broader psychosocial dysfunction. For example, within a sample of depressed persons, those who reported the most similar reactions in sad and neutral contexts (the pattern predicted by ECI) were found to exhibit the highest depression severity, to have been depressed for the longest period of time, and to have the lowest levels of overall psychosocial functioning (Rottenberg, Kasch, et al., 2002). Similarly, ECI has also been shown to predict prospective depression-related impairment: Those depressed individuals who displayed the most similar behavioral and heart-rate reactions in amusing and neutral contexts were the least likely to recover 6 months later.

Toward a More Comprehensive Test of the ECI Hypothesis

The ECI hypothesis is rooted in a functional perspective on emotion that conceives of emotional reactions as adaptive responses to challenges and opportunities. In this view, depression involves a global disruption of these responses as part of a larger pattern of cessation of action and withdrawal. A number of findings now suggest that the ECI hypothesis may provide an accurate and clinically useful account of emotional reactivity in MDD. At the same time, the ECI hypothesis has received remarkably few explicit tests. Consequently, three basic aspects of ECI are poorly understood and are in need of clarification: (a) the conditions under which it holds, (b) its clinical significance, and (c) the emotional response domains to which it applies.

Under What Conditions Does ECI hold?

Experimental studies of emotional reactivity in MDD have used a small number of procedures to elicit emotion, most often involving normative stimuli presented via an external medium, such as films or slides. On the one hand, these procedures provide investigators with reliable, standardized probes of emotional reactivity. On the other hand, the use of external, normatively derived stimulus sets raises the concern that these experimental paradigms may not constitute a robust test of ECI. Existing findings of ECI may be driven by the experimental paradigm and may be subject to methodological explanations. For instance, depressed individuals might find it difficult to engage with external stimuli because of MDD-related impairments (e.g., concentration difficulties, self-focus) or might not react to normative stimuli with themes that are largely irrelevant to persons suffering from a serious disorder.

Cognitive theories of MDD suggest that idiographic, person-specific themes are particularly important for conducting a robust test of ECI. One influential typology, for example, has identified two clusters of cognitive schemas that are activated differentially by person-specific content regarding either social relationships or achievement (Beck, 1983; Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982). Therefore, if depressed persons' negative schemas are organized around idiographic, person-specific content, depressed individuals should tend to respond strongly to idiographic

negative content (Lazarus, 1991). In other words, experiments using idiographic stimuli would appear to be propitious for the alternative negative potentiation hypothesis, thus posing a more conservative test of ECI.

How Does ECI Relate to Depression Vulnerability?

Because most existing observations of emotional reactivity are taken from individuals who are currently depressed, the relation between emotional reactivity and vulnerability to future depression is unclear. For example, if ECI contributes to depression onset, it should have a "trait-like" quality and should be evident among formerly depressed individuals when they are currently euthymic. Thus, currently and formerly depressed individuals should exhibit similar patterns of emotional responding, and both of these groups of participants should differ from healthy nonpsychiatric control participants. Conversely, if ECI is a marker of a depressed mood state, one would expect to see this pattern of emotional reactivity *only* among currently depressed individuals.

Consistent with the possibility that ECI is traitlike, Iacono et al. (1984) found that currently and formerly depressed participants exhibited similarly attenuated electrodermal responding across both emotional and nonemotional stimuli relative to control participants. More recently, Dichter et al. (2004) found that before antidepressant treatment, depressed persons' emotion-startle modulation was attenuated relative to control participants and remained attenuated even after symptomatic improvement. Finally, Sigmon and Nelson-Gray (1992) found that both formerly depressed and currently depressed persons were characterized by potentiated electrodermal responding to negative stimuli relative to healthy control participants. Though these findings were opposite of ECI in direction, it is important to note that the formerly and currently depressed participants exhibited the same pattern of emotional reactivity. In sum, although available data are limited, they suggest that it is more likely that ECI is more traitlike than a mood-state-dependent pattern of emotional reactivity.

Is ECI in Depression Observed Across All Domains of Emotional Reactivity?

Emotional reactivity involves changes in a number of response domains, perhaps most notably in subjective experience, behavior, and physiology. It is not clear whether ECI in MDD operates equally across all of these domains. For example, some findings suggest that abnormalities consistent with ECI are strong in the behavioral domain but are not apparent in emotional experience (e.g., Berenbaum & Oltmanns, 1992; Gehricke & Shapiro, 2000). In contrast, other findings suggest that ECI abnormalities are strong in the experiential domain but are not evident in the behavioral and physiological domains (Rottenberg, Kasch, et al., 2002; Sloan et al., 1997). These discrepancies across studies may reflect the heterogeneity of depression (Beckham, Leber, & Youll, 1995), the loose coupling among domains of emotional functioning that is observed either generally (Lang, 1978) or specifically in depressed persons (Brown, Schwartz, & Sweeney, 1978), or simply the wide variation in test conditions and methodologies used to elicit and assess emotion. To address this issue, researchers need to use careful multimethod assessments of emotional reactivity during well-controlled conditions.

Present Study

The present study was designed to test the ECI hypothesis against competing views of emotion in MDD (positive attenuation and negative potentiation). To test the ECI hypothesis under a broad range of conditions, we used a comprehensive stimulus battery that focused on happy, neutral, and sad states. To provide a conservative test of ECI, we varied the dimension of personal concern and included idiographic stimuli, in addition to stimuli that were derived from normative themes. We further addressed the generalizability of ECI by presenting these stimuli in both external (films) and internal (imagery) modes. To examine whether ECI might represent a trait-like pattern of response, we compared emotional reactivity in a group of currently depressed individuals with reactivity in groups of formerly depressed individuals and healthy control participants who had never been depressed. Finally, to examine whether ECI is observed across different domains of emotional responding, we used a multimethod strategy to assess emotional reactivity, obtaining measures of emotional experience, emotional behavior, and autonomic physiology for each stimulus condition.

On the basis of our review of the literature, our primary hypothesis was that depressed persons would exhibit ECI relative to healthy participants. Also on the basis of prior work, we expected that ECI would be trait-like. Hence, formerly depressed persons were also expected to exhibit ECI relative to healthy participants. Finally, we anticipated that ECI effects would hold across domains of emotional reactivity (e.g., emotion experience, behavior, and autonomic physiology), although on the basis of our own work (Rottenberg, Kasch, et al., 2002), we also expected that ECI would be most pronounced in self-reports of emotion experience.

Method

Overview of Design

In an initial session, participants were diagnosed and videotaped during an interview in which they provided emotionally neutral demographic information as well as emotionally charged information about their peak lifetime happy and sad events. These happy, sad, and neutral videotaped segments were used to create the idiographic films used in the psychophysiology session alongside the standardized (normative) valenced films. The subsequent psychophysiology session assessed emotional reactivity using a 3 (emotion: happy, neutral, sad) \times 2 (type: normative, idiographic) \times 2 (presentation mode: external [viewing], internal [imagining]) within-subjects design. Data analysis focused on group comparisons of self-reported experiential, behavioral, and physiological measures of emotional reactivity to valenced normative and idiographic stimuli.

Clinical Assessments

Participant Screening

Participants were recruited from a variety of sources; clinical participants were solicited from two outpatient psychiatry clinics in a university teaching hospital, as well as through advertisements posted in numerous locations within the local community (e.g., Internet bulletin boards, university kiosks, supermarkets). Healthy control participants were recruited from the community through advertisements posted in the same locations. Participants' responses to a telephone interview provided initial selection information. This phone screen established that participants were fluent in English and were between 18 and 60 years of age. As per the requirements

of a larger parent project, participants were excluded for reported lifetime history of brain injury or primary psychotic symptoms, lifetime diagnoses of bipolar disorder, current diagnoses of panic disorder or social phobia, behavioral indications of impaired mental status or mental retardation, or reported signs of alcohol or substance dependence or abuse within the past 6 months. More important, this interview was used to identify individuals who were likely to meet criteria for one of three groups: (a) currently depressed individuals; (b) formerly depressed individuals; and (c) never-depressed healthy control participants.

A total of 660 individuals were screened by telephone. Of this group, 158 met initial eligibility criteria and came to the laboratory for an in-person interview with the Structured Clinical Interview for *DSM-IV* Axis I (SCID-I; First, Gibbons, Spitzer, & Williams, 1995). The SCID-I was used to confirm that study entry criteria were met. Healthy control participants were interviewed to exclude those with lifetime diagnoses of any Axis I disorders. Potential control participants were excluded from the study on the basis of the same general and medical criteria that were used for the clinical participants. Of potentially eligible participants, 80 (51%) met all the entry criteria during the interview with the SCID-I. Finally, 13 individuals who passed the SCID-I did not complete psychophysiology sessions for a variety of reasons including scheduling problems, unwillingness to continue with the study, or equipment failure.

The final participant sample consisted of 19 unipolar depressed persons (73.7% female), 22 recovered unipolar depressed persons (68.2% female), and 26 nondepressed control participants (69.2% female). Forty-two percent of the currently depressed group and 14% of the recovered depressed group were receiving some form of psychological treatment. Thirteen participants were receiving pharmacotherapy (32% of the currently depressed group, 32% of the recovered depressed group).¹ All participants provided written informed consent prior to the experimental session and were paid \$25/hr.

Clinical Diagnoses

All currently depressed participants met *DSM-IV* criteria (APA, 1994) for current MDD, according to the SCID-I. SCID-I interviewers had previous experience with administering structured clinical interviews and were trained specifically to administer the SCID-I interview. In prior work, this team of interviewers achieved excellent interrater reliability for MDD ($\kappa = 1.00$; Rottenberg, Kasch, et al., 2002).

All formerly depressed participants met SCID-I criteria for a past episode of MDD. In addition, guidelines from the National Institute of Mental Health Collaborative Program on the Psychobiology of Depression were implemented to screen out individuals who had current symptoms of depression (e.g., Keller et al., 1992). These guidelines required that, to be considered fully recovered, respondents must have reported virtually no signs of depressive illness (e.g., no more than two symptoms, and those symptoms experienced only to a mild degree) when questioned week-by-week using a modified version of the SCID-I about the presence of all nine *DSM-IV* depression symptoms during the 8 weeks prior to the interview. We adopted this stringent definition of recovery so that the formerly depressed participants would be relatively free of the significant functional impairment known to be associated with residual depressive symptoms (Judd, Paulus, Wells, & Rapaport, 1996). Therefore, formerly depressed participants who exhibited current subsyndromal or syndromal depression were excluded from the sample.

¹ We found no differences in emotional reactivity as a function of medication status. The pattern of effects was unchanged when medication status (medicated vs. unmedicated) was used as a covariate.

Measures of Clinical Functioning

Depression and Anxiety

The Beck Depression Inventory (BDI; Beck et al., 1979) and the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) were administered to measure self-reported depression severity and self-reported anxiety severity. Both inventories are 21-item, self-report measures of the intensity of depression or anxiety. Items on the BDI and BAI assess cognitive, affective, behavioral, and physiological symptoms of depression and anxiety, with the total score representing a combination of the number of symptom categories endorsed and the severity of the particular symptoms. The acceptable reliability and validity of these measures have been well documented (Beck, Epstein, et al., 1988; Beck, Steer, & Garbin, 1988).

Length of Current Depressive Episode

In conjunction with the participants, the SCID-I interviewer determined the onset of the current depressive episode for all individuals diagnosed with current depression.

Global Functioning

The Global Assessment of Functioning Scale (GAF; Axis V, *DSM-IV*; APA, 1994) was used to assess global functioning. The GAF is a single rating scale used to evaluate a person's overall level of psychological, social, and occupational functioning. Ratings are made on the basis of the SCID-I interview and range from 1 (lowest level of functioning) to 100 (highest level of functioning). The reliability of the GAF has been demonstrated in prior work (Endicott, Spitzer, Fleiss, & Cohen, 1976) as well as with this team of interviewers (Kasch, Rottenberg, Arnow, & Gotlib, 2002; Rottenberg, Kasch, et al., 2002).

Psychophysiological Assessment

Stimuli

Four stimulus categories were derived from the crossing of the personal concern dimension (normative, idiographic) with two presentation modes (external, internal). Stimuli were displayed on a 20-in. TV monitor at a viewing distance of 1.75 m.

Normative external. Films are dynamic visual stimuli that have been shown to elicit emotions ethically and reliably with relatively low demand characteristics. Happy, sad, and neutral normative films were used, with film selection based on criteria recommended by Rottenberg, Ray, and Gross (in press). The happy film involved themes of fulfillment and connectedness to others. The film was 237-s long and depicted a boy overcoming the challenge of learning to ride a bike with his father's help, followed by scenes in which the boy discovers his parents playfully dancing together. The sad film involved themes of loss and abandonment. It was 170-s long and depicted a death scene in which a boy becomes distraught at the death of his father. This film elicits reports of sadness and observable sadness behaviors in nondepressed individuals (Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002). The neutral film lasted 180 s and depicted coastal landscape scenery. This was selected because it fully engages participants' attention, induces little report of emotion or emotion-expressive behavior in nondepressed participants, and is well-tolerated by participants (Rottenberg, Kasch, et al., 2002).

Normative internal. Emotional imagery has been shown to elicit psychophysiological responses that are similar in nature to those of external emotional stimuli (e.g., Lang, 1979). In the current study, the content of normative emotional imagery was keyed to the content of each standardized film. That is, after each film, participants were instructed to hold the

just-viewed film in mind and to focus on its visual, auditory, and emotional qualities. Mental imagery trials lasted 180 s.

Idiographic external. One challenge in conducting research with idiographic emotional stimuli is finding a procedure to reliably generate personally meaningful emotional events. Because procedures in prior studies of MDD have been relatively informal (e.g., Greden et al., 1986; Schwartz et al., 1976), for the present study we developed an Emotions Interview to generate idiographic stimuli. The Emotions Interview is a brief (~15 min) semistructured interview designed to elicit details concerning the maximally happy and sad life moments experienced by each participant. An initial instructional set requests participants to describe their happy and sad life events, "in enough detail so a person who was not there could understand why this moment was significant for you." Interviewers ask additional questions to help participants situate their emotional events in time and place (e.g., "Who was around at the time?"). Once the context of the emotional events was established, interviewers used standardized probes to elicit the personal meaning and emotional qualities of the participant's life event (e.g., "Of all of the events from your life that you could have chosen as the happiest/saddest, why did you choose this one?"). A final module elicited basic demographic information from participants. This module was used as a neutral comparison condition. All Emotions Interviews were videotaped. Three idiographic stimulus films were generated for each participant using the first 180 s videotaped from the happy, sad, and neutral modules.

Idiographic internal. Idiographic mental imagery trials were identical in all ways to the normative imagery trials, except that they followed idiographic films.

Procedure

Participants were greeted and positioned in a comfortable chair facing a video monitor in a quiet, well-furnished laboratory room. After procedures for connecting physiological monitoring devices were explained and monitors were attached, participants were told that they would be asked to view short films and to use their "mind's eye" to visualize certain types of scenes. Presentation order of stimulus type (normative, idiographic) and emotion conditions (neutral, happy, sad) were counterbalanced across participants. Presentation mode (external, internal) took place in a fixed order, with imagery following films, to allow the content of each imagery trial to be yoked to the preceding film. Before each film-imagery block, participants sat quietly for 60 s with their eyes open. A current emotional state questionnaire was administered at the start of each baseline and immediately after each experimental task. In order to reduce carryover effects, we ensured that each film-imagery block was followed by a 60 s filler task in which participants copied three geometric figures. In pilot work, participants reported this task to be moderately engaging and relatively undemanding.

For film trials, participants were instructed to simply watch carefully. For imagery trials, participants were asked to form "a vivid picture" of the just-viewed film in their mind and to close their eyes and to "focus on the sights, the sounds, and the emotions that this film brings to mind." Imagery trials began once participants indicated comprehension of the task and readiness to begin. At the end of these procedures, the experimenter debriefed the participant and probed for evidence of continued dysphoria. Participants were then disconnected from monitoring devices and paid.

Measures

Self-Report Assessment of Emotion

Self-reported affective state was sampled with a brief questionnaire administered at baseline and after each experimental task. Participants reported on the following states: happiness, amusement, sadness, and anxiety. Hypotheses concerned happiness and sadness, the states targeted by the manipulation. Participants also rated each task for personal rele-

vance and indicated their ability to carry out task instructions. All items were rated on a 9-point unipolar scale, ranging from 0 = *not at all* to 8 = *extremely*.

Emotional Behavior

EMG was used in the present study to assess emotional behavior. Electrical activity was recorded directly from the facial muscles via electrodes applied to the surface of the skin from two facial muscle groups that are associated with happy and sad expressions (e.g., Larsen, Norris, & Cacioppo, 2003): (a) the zygomatic muscles, which are located between the angles of the mouth and cheekbones bilaterally and are associated primarily with smiling, and (b) the corrugator muscles, which are located bilaterally above the bridge of the nose and are involved in the expression of sadness, grief, anger, and pain. One advantage of using EMG to record facial behavior is that it is sensitive to subtle changes in facial tonus that observers cannot accurately assess. Thus, EMG here served as a measure of expressive behavior rather than being treated as a physiological measure. Procedures for attaching EMG sensors, as well as recording and signal processing, followed guidelines recommended by Fridlund and Cacioppo (1986). EMG data were recorded using a bandpass filter from 10 to 500 Hz and were sampled at 1000 Hz. Zygomatic and corrugator EMG signals were expressed in microvolts.

Physiological Measures

Four primary physiological measures were obtained. These measures were selected to sample from major organ systems known to be important in emotional responding (cardiac, vascular, electrodermal, and respiratory) to allow for continuous measurement and to be as unobtrusive as possible. The measures were as follows.

1. *Heart rate*: Beckman miniature electrodes were placed in a bipolar configuration on opposite sides of the participant's chest. The interbeat interval was calculated as the interval (in milliseconds) between successive R waves in the electrocardiogram (ECG) and converted to instantaneous heart rate.
2. *Pulse transmission time to the finger*: A UFI photoplethysmograph recorded blood volume in the finger using a photocell taped to the distal phalange of the second finger of the nondominant hand. The time interval (in milliseconds) was measured between the R wave of the ECG and the upstroke of the peripheral pulse at the finger site.
3. *Skin conductance response rate*: A constant-voltage device passed a small voltage between Beckman regular electrodes attached to the palmar surface of the proximal phalanges of the first and second fingers of the nondominant hand. Skin conductance fluctuations were detected as changes in skin conductance level from a zero-slope baseline exceeding 0.02μ Siemens.
4. *Respiratory rate*: two bands (Respirace) were placed around the upper thorax and abdomen and connected to an inductive plethysmography system (Ambulatory Monitoring, Ardsley, NY). These four primary physiological measures were sampled at 400 Hz.

Data Reduction

Behavioral and physiological data were reduced off-line, using custom MATLAB software (Wilhelm, Grossman, & Roth, 1999) to extract segments of raw data and then perform waveform transformation, feature detection, and graphic display for each channel. A 60 Hz digital notch filter was used to attenuate ambient electronic noise. Artifacts were edited

manually for each channel. Period averages were calculated for each experimental epoch. Because untransformed EMG voltages are not normally distributed, EMG data for each condition were converted into percentage change from baseline scores. For this computation, the 60-s quiet sitting period before each film–imagery block was used as a baseline.

Hypothesis Testing

Given the relatively loose coupling of emotional response domains (Lang, 1978) and our specific interest in the relation between ECI and response domain, we organized hypothesis testing around each domain of response (i.e., self-reported experience, behavior, physiology). Omnibus tests in the self-report and behavioral domains used univariate repeated-measures analyses of variance (ANOVAs) to permit separate examination of indicator variables for happiness and sadness (e.g., zygomatic vs. corrugator EMG; Larsen et al., 2003). Omnibus tests of the physiological domain used multivariate repeated-measures ANOVAs. In these analyses, group (current MDD, former MDD, healthy control) was a between-subjects factor, and emotion condition (neutral, sad, happy) and stimulus type (normative, idiographic) were within-subjects factors. We also included a presentation mode factor (external, internal) as a within-subjects factor to further bolster the generalizability of our study. Initial analyses, however, revealed very few effects of presentation mode. In only 1 of 10 of these analyses was mode significant (a nonhypothesized three-way interaction of group, type, and presentation mode for zygomatic EMG). Consequently, we collapsed across the mode factor in subsequent analyses to increase our statistical power.²

Our primary hypothesis was that currently depressed individuals would be characterized by ECI. This hypothesis was tested in three steps. First, it was expected that the emotion effect would be significant in the omnibus analysis, indicating a successful manipulation of emotional valence. Second, it was expected that the Group \times Emotion interaction effect would be significant, indicating that emotion modulation differed by group. For Group \times Emotion interactions, emotion modulation was examined separately in each group. If ECI held, emotion modulation would be present in the healthy control participants but absent in the currently depressed group. In addition, we also predicted that ECI would exhibit trait-like features and would be independent of current mood state. Thus, we anticipated that on those variables for which currently depressed participants exhibited ECI, formerly depressed persons would also fail to exhibit emotion modulation. A positive result would indicate likely state independence (i.e., an effect common to both groups of depression-vulnerable persons), whereas a negative result on this test would indicate state dependence (i.e., an effect unique to current depression).

Results

Demographic and Clinical Characteristics

As presented in Table 1, the three diagnostic groups were equivalent with respect to gender, ethnic composition, marital status, age, income, and education level (all $ps > .1$). Consistent with the criteria used in creating the three groups, the groups differed in self-reported severity of depression and anxiety: BDI, $F(2, 64) = 99.37$; BAI, $F(2, 65) = 51.62$, both $ps < .001$, with currently depressed participants obtaining higher scores on the BDI and BAI than did both formerly depressed participants and

² Exploratory analyses also included gender and experimental condition as between-subjects factors. There were no main effects or interactions of these two variables, and these factors were collapsed in subsequent analyses.

Table 1
Demographic and Clinical Characteristics of the Depressed,
Formerly Depressed, and Never-Depressed (Control)
Participants

Characteristic	Group		
	Depressed (<i>n</i> = 19)	Recovered (<i>n</i> = 22)	Control (<i>n</i> = 26)
Gender, female: <i>N</i> (%)	14 (73.7)	15 (68.2)	18 (69.2)
% married	27.7	22.3	28.0
% Caucasian	84.2	77.3	57.7
Age: <i>M</i> (<i>SD</i>)	35.7 (7.5)	33.7 (9.3)	33.6 (10.7)
Education: <i>M</i> (<i>SD</i>)	6.9 (1.3)	6.7 (1.6)	6.5 (1.5)
Annual income	3.9 (1.9)	4.0 (1.4)	4.3 (1.3)
BDI score	24.9 (8.4) _a	5.3 (4.8) _b	3.0 (3.0) _b
BAI score	19.2 (9.7) _a	3.5 (3.7) _b	2.7 (2.8) _b
GAF score	53.2 (4.5) _a	83.5 (5.6) _b	88.0 (3.7) _c
MDD length (mo)	6.7 (5.6)		

Note. Different subscripts within rows indicate significant group differences. Education was assessed on an 8-point scale, with higher numbers representing more education—a score of 6.7 reflects some college education. Income was assessed on a 6-point scale, with higher numbers representing more income—a score of 4.0 reflects annual income between \$50,000 and \$75,000. BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; GAF = Global Assessment of Functioning Scale; MDD = major depressive disorder; mo = months.

healthy control participants, who did not differ from one another. As expected, the groups also differed with respect to their GAF scores, $F(2, 62) = 330.29, p < .001$. The healthy control participants had the highest GAF scores, followed by the formerly depressed group, followed by the currently depressed group, who had the lowest functioning (all pairwise differences, $p < .001$).

Self-Reported Emotion Experience

Happiness

Mean levels of reported happiness for normative and idiographic stimuli by diagnostic group are presented in Table 2. A three-way (Group \times Emotion \times Stimulus Type) ANOVA yielded

Table 2
Mean Reported (*SE*) Happiness Experience by Group, Emotion,
and Stimulus Type

Emotion and stimulus type	Group		
	Depressed	Recovered	Control
Happy			
Normative ^a	3.26 (0.35)	5.02 (0.26)	5.42 (0.26)
Idiographic ^a	2.44 (0.37)	5.00 (0.25)	5.56 (0.28)
Neutral			
Normative ^a	3.10 (0.39)	4.54 (0.34)	4.90 (0.31)
Idiographic ^a	1.81 (0.36)	4.16 (0.35)	4.60 (0.34)
Sad			
Normative ^b	1.97 (0.39)	2.68 (0.35)	3.40 (0.31)
Idiographic ^a	1.28 (0.33)	3.36 (0.40)	3.81 (0.36)

^a Control participants, recovered participants $>$ depressed participants, $p < .01$.

^b Control participants $>$ depressed participants, $p < .01$.

main effects for emotion condition, $F(2, 60) = 49.5, p < .001$, and group, $F(2, 61) = 17.72, p < .001$. Consistent with the intended valence manipulation, follow-up tests revealed that participants reported higher levels of happiness in the happiness conditions ($M = 4.48, SE = .16$) than in the sad conditions ($M = 2.77, SE = .19$), with the neutral conditions falling between the two valenced conditions ($M = 3.86, SE = .18$) and significantly differing from both (all pairwise differences, $p < .001$). The main effect for group reflected the finding that currently depressed individuals reported less happiness ($M = 2.38, SE = .29$) than did healthy control participants ($M = 4.61, SE = .25$) and formerly depressed individuals ($M = 4.13, SE = .26$), both $ps < .001$. As shown in Table 2, this pattern of differences held in all of the experimental conditions. The predicted interaction of group and emotion condition was nonsignificant, $F(4, 122) < 1$.

There was, however, an interaction of group and stimulus type, $F(2, 60) = 8.16, p = .001$. To decompose this interaction, we conducted separately paired *t* tests examining the effect of stimulus type for each diagnostic group. These follow-up analyses indicated that currently depressed individuals reported higher levels of happiness to normative stimuli ($M = 2.84, SE = .35$) than to idiographic stimuli ($M = 1.92, SE = .31$), $t(17) = 3.95, p = .001$, whereas participants in the other two diagnostic groups did not distinguish between stimulus types in their happiness reports (both $ps > .10$).³

Sadness

Mean levels of reported sadness for normative and idiographic stimuli by diagnostic group are presented in Table 3. The three-way (Group \times Emotion \times Stimulus Type) ANOVA yielded an effect of emotion condition, $F(2, 61) = 34.39, p < .001$. Consistent with the intended valence manipulation, post hoc tests revealed that participants reported higher levels of sadness in the sadness conditions ($M = 3.62, SE = .16$) than in the happy conditions ($M = 2.05, SE = .17$), with the neutral conditions falling in between ($M = 2.26, SE = .19$) and differing from both of the emotionally valenced conditions (all differences, $p < .001$). The Emotion \times Group interaction predicted by Hypothesis 1 (ECI) was significant, $F(4, 124) = 2.87, p < .05$. To decompose this interaction, we conducted separate follow-up ANOVAs for each

³ There were Emotion \times Stimulus Type interactions for happiness, $F(2, 60) = 7.69, p = .001$, and for sadness, $F(2, 61) = 7.00, p < .005$. For happiness, comparisons between idiographic and normative stimuli separately for each emotion indicated that this interaction was due to the fact that during the neutral conditions, normative stimuli were associated with higher levels of reported happiness than were idiographic stimuli, $t(64) = 3.28, p < .01$, a pattern not observed in either of the emotionally valenced conditions. For sadness, comparisons between idiographic and normative stimuli separately for each emotion condition indicated that this interaction was due to the fact that during the neutral conditions, idiographic stimuli received higher levels of reported sadness than did normative stimuli, $t(65) = 3.40, p = .001$, a pattern not observed in either of the emotionally valenced conditions. In other words, exposure to landscape scenery stimuli appeared to yield a somewhat more positive affective state in participants than did the neutral module of the relived emotion interview. Although this pattern did not threaten our hypotheses, the fact that the neutral conditions were not matched on experience reports should be considered a limitation of the study.

Table 3
Mean Reported (SE) Sadness Experience for Normative and Idiographic Stimuli by Group

Emotion and stimulus type	Group		
	Depressed	Recovered	Control
Happy			
Normative ^a	3.87 (0.47)	1.20 (0.31)	0.60 (0.18)
Idiographic ^a	4.34 (0.40)	1.20 (0.28)	0.73 (0.23)
Neutral			
Normative ^a	3.71 (0.38)	1.09 (0.28)	0.92 (0.22)
Idiographic ^a	4.37 (0.45)	1.64 (0.39)	1.40 (0.34)
Sad			
Normative ^b	4.72 (0.34)	3.13 (0.42)	3.34 (0.44)
Idiographic ^a	4.97 (0.48)	2.93 (0.34)	2.17 (0.36)

^a Depressed participants > control, recovered participants, $p < .01$.

^b Depressed > control, recovered participants, $p < .05$.

diagnostic group. The healthy control group exhibited the expected valence modulation for sadness reporting, $F(2, 23) = 26.19, p < .001$. Consistent with ECI, however, currently depressed individuals did not discriminate in their sadness reports as a function of stimulus valence, $F(2, 16) = 2.53, p > .1$. However, inconsistent with our expectation that ECI would be trait-like, the formerly depressed group resembled healthy control participants, also exhibiting clear valence modulation for sadness reporting, $F(2, 20) = 14.32, p < .001$.

The uniqueness of this pattern of ECI to the depressed group was related to the fact that depressed individuals reported remarkably high levels of sadness during the neutral and happy conditions, contexts in which the report of sadness would ordinarily be very low. Table 3 shows that unlike participants in the two other groups, currently depressed participants reported considerable sadness across all conditions. This main effect of group, $F(2, 62) = 36.05, p < .001$, was robust, with currently depressed individuals' overall sadness reports being higher ($M = 4.48, SE = .24$) than were the sadness reports of both never-depressed control participants ($M = 1.58, SE = .24$) and formerly depressed individuals ($M = 1.87, SE = .25$; both $ps < .001$).

Finally, as was seen with the happiness data, the Group \times Type interaction for sadness approached significance, $F(2, 62) = 2.36, p = .10$. Paired t tests conducted within each group indicated that currently depressed individuals tended to report higher levels of sadness to idiographic than to normative stimuli—depressed paired t test for stimulus type, $t(17) = 1.85, p = .08$ —whereas participants in the other two groups did not discriminate between stimulus types (both $ts < 1$).

In sum, currently but not formerly depressed individuals exhibited ECI for sadness, and currently depressed individuals reported less happiness than did the other diagnostic groups across all contexts. It is interesting to note that only individuals with current MDD distinguished between normative and idiographic emotional stimuli, reporting lower levels of happiness and higher levels of sadness to idiographic stimuli.

Emotion-Expressive Behavior

Zygomatic EMG

Zygomatic EMG data for each diagnostic group and experimental condition are reported in Table 4. The three-way (Group \times

Table 4
Mean Percentage Change (SE) of Zygomatic EMG by Condition and Group

Emotion and stimulus type	Group		
	Depressed	Recovered	Control
Happy			
Normative*	14.4 (9.0)	9.9 (8.0)	-6.5 (4.0)
Idiographic	9.0 (6.0)	7.5 (5.0)	0.9 (4.0)
Neutral			
Normative	1.1 (5.0)	-7.2 (4.0)	-7.6 (3.0)
Idiographic	5.3 (4.0)	-2.2 (4.0)	0.5 (3.0)
Sad			
Normative	3.7 (3.0)	-1.2 (3.0)	0.8 (5.0)
Idiographic	2.6 (6.0)	-5.6 (4.0)	6.1 (4.0)

Note. EMG = electromyography.

* Depressed participants > control participants, $p < .05$.

Emotion \times Stimulus Type) ANOVA on zygomatic activity yielded a main effect of emotion, $F(2, 55) = 5.29, p < .01$. Consistent with the intended valence manipulation, zygomatic activity was higher in the happy condition than in both the neutral and the sad conditions. Consistent with our primary hypothesis, the interaction of emotion and group was significant, $F(4, 112) = 2.67, p < .05$. To decompose this interaction, we conducted analyses of the emotion effect separately for each diagnostic group. Inconsistent with our expectation, the healthy control participants did not exhibit an effect for emotion nor did the current depressed group (both $ps > .15$). The emotion effect was evident only in the formerly depressed group, $F(2, 16) = 3.45, p < .06$, reflecting the fact that formerly depressed individuals exhibited higher levels of zygomatic activity in the happy condition relative to the neutral and sad conditions. This finding was again inconsistent with our expectation of similar patterns of emotional reactivity in currently and formerly depressed persons.

Corrugator EMG

Table 5 presents mean corrugator levels for normative and idiographic stimuli by diagnostic group. A three-way (Group \times

Table 5
Mean Percentage Change (SE) Corrugator EMG by Condition and Group

Emotion and stimulus type	Group		
	Depressed	Recovered	Control
Happy			
Normative	28.0 (18.0)	18.6 (12.0)	7.6 (5.0)
Idiographic	-0.7 (4.0)	0.5 (7.0)	1.8 (5.0)
Neutral			
Normative	0.5 (7.0)	21.0 (9.0)	6.3 (5.0)
Idiographic	7.6 (7.0)	12.0 (9.0)	12.1 (5.0)
Sad			
Normative	27.0 (12.0)	37.1 (15.0)	16.4 (8.0)
Idiographic ^a	0.2 (6.0)	18.4 (7.0)	6.1 (4.0)

Note. EMG = electromyography.

^a Recovered participants > depressed participants, $p < .05$.

Emotion × Stimulus Type) ANOVA conducted on corrugator responding yielded a main effect of emotion, $F(2, 55) = 3.51, p < .01$. Consistent with the intended valence manipulation, corrugator levels were higher in the sad condition than in the neutral or happy conditions. The Emotion × Group effect predicted in our primary hypothesis, however, was nonsignificant.

Autonomic Physiology

Autonomic data for normative and idiographic stimuli by diagnostic group are presented in Table 6. The three-way (Group × Emotion × Stimulus Type) multivariate analysis of variance (MANOVA) on autonomic variables revealed a nonsignificant effect of emotion, $F(2, 47) < 1$. Because there was an interaction of emotion and stimulus type, $F = (2, 47) = 9.76, p < .001$, we proceeded to examine the valence modulation component of the ECI hypothesis. To determine the source of this interaction, we conducted separate Emotion × Stimulus Type ANOVAs for each of the autonomic measures. This effect was significant for respiratory rate, $F = (2, 62) = 21.61, p < .001$, and skin conductance response rate, $F = (2, 54) = 13.54, p < .001$, but not for heart rate, $F = (2, 61) = 1.79, p > .1$, or finger pulse transit time, $F < 1$.

For respiratory rate, the effect of emotion was significant for normative stimuli, $F(2, 62) = 46.51, p < .001$, but not for idiographic stimuli, $F(2, 62) < 1$. A similar pattern emerged for skin conductance response rate: The effect of emotion was significant for normative stimuli, $F(2, 57) = 25.09, p < .001$, but not for

idiographic stimuli $F(2, 58) = 1.16, p > .1$. For respiratory rate, the emotion effect for normative stimuli was due to the fact that respiratory rate was highest for happy stimuli ($M = 16.61, SE = .35$), next highest for sad stimuli ($M = 15.96, SE = .39$), and lowest for neutral stimuli ($M = 15.00, SE = .36$), all pairwise differences, $p < .05$. Similarly, for skin conductance response rate, the emotion effect for normative stimuli was driven by participants' greater responding during the emotionally valenced conditions relative to the neutral conditions (sad $M = 3.77, SE = .43$; happy $M = 3.77, SE = .40$; neutral $M = 2.41, SE = .34$).

In sum, consistent with the intended valence manipulation, the data for normative stimuli indicate that greater autonomic responses were observed during exposure to emotional material than to neutral material. This expected valence effect was absent for idiographic stimuli, perhaps as a function of the greater heterogeneity of these stimuli. Although the expected pattern of valence modulation was exhibited for normative stimuli, the Emotion × Group interaction predicted by our primary hypothesis was nonsignificant.

Secondary Analyses: Personal Relevance and Task Engagement

Prior ECI-relevant findings using normative stimuli presented via an external medium are subject to alternative explanations involving stimulus irrelevance and low task engagement. In addition to addressing these concerns by incorporating idiographic and

Table 6
Autonomic Variables (SE) for Normative and Idiographic Stimuli by Group

Variable	Group		
	Depressed	Recovered	Control
Heart rate (beats/min)			
Happy norm	76.76 (2.79)	75.43 (2.04)	72.27 (2.45)
Happy idio	76.12 (2.80)	74.98 (2.04)	71.36 (2.50)
Neutral norm	75.57 (2.77)	75.48 (2.12)	71.73 (2.43)
Neutral idio	75.79 (2.74)	75.24 (2.04)	72.1 (2.64)
Sad norm	73.13 (2.77)	75.30 (2.30)	71.59 (2.52)
Sad idio	75.90 (2.63)	75.40 (2.04)	72.26 (2.64)
Finger pulse transit time (ms)			
Happy norm	250.5 (3.9)	248.5 (3.8)	256.1 (4.2)
Happy idio	247.6 (3.4)	247.4 (3.4)	254.6 (3.6)
Neutral norm	252.6 (3.3)	249.7 (3.8)	256.8 (4.2)
Neutral idio	249.5 (3.5)	249.0 (3.8)	255.8 (4.0)
Sad norm	250.4 (4.0)	249.6 (3.9)	257.1 (4.5)
Sad idio	248.8 (3.8)	246.7 (3.7)	254.7 (4.2)
Respiration rate (breaths/min)			
Happy norm	16.81 (0.61)	17.20 (0.54)	15.94 (0.63)
Happy idio	15.91 (0.62)	17.00 (0.53)	15.82 (0.60)
Neutral norm	15.06 (0.62)	15.31 (0.55)	14.57 (0.64)
Neutral idio	16.11 (0.56)	16.73 (0.55)	16.05 (0.63)
Sad norm	15.42 (0.79)	17.02 (0.55)	15.35 (0.61)
Sad idio	15.68 (0.67)	16.71 (0.51)	16.04 (0.56)
Skin conductance response rate (responses/min)			
Happy norm	4.39 (0.66)	3.65 (0.74)	3.44 (0.60)
Happy idio	4.76 (0.76)	4.24 (0.75)	4.27 (0.55)
Neutral norm	2.88 (0.66)	2.51 (0.61)	1.99 (0.41)
Neutral idio	5.03 (0.90)	3.65 (0.66)	4.33 (0.74)
Sad norm	3.97 (0.75)	3.49 (0.59)	3.62 (0.80)
Sad idio	5.31 (0.90)	3.88 (0.69)	4.73 (0.74)

Note. norm = normative; idio = idiographic.

internally generated stimuli into the design of this study, we also conducted secondary analyses of participants' ratings of stimulus relevance and task engagement for evidence that they might account for observed effects.

Personal Relevance

Group had no effect on ratings of personal relevance across all categories of stimuli, $F(2, 62) < 1$, a finding that is inconsistent with a stimulus irrelevance account of ECI. Group did interact with emotion condition, however, $F(4, 124) = 3.07, p < .05$. Healthy control participants and formerly depressed individuals rated sad stimuli as being less personally relevant than neutral and happy stimuli: control participants, $F(2, 23) = 6.49, p < .01$; formerly depressed participants, $F(2, 19) = 9.98, p = .001$. In contrast, currently depressed participants found all of the emotion conditions equally relevant, $F(2, 17) < 1$. In sum, it is difficult for these personal relevance data to explain currently depressed persons' ECI for sadness reports or their generally low levels of reported happiness.

Finally, and not surprising, across all groups of participants, idiographic stimuli were rated as being more personally relevant than normative stimuli, $F(1, 62) = 215.77, p < .001$. Because all diagnostic groups exhibited this effect, it is unlikely that the greater personal relevance of idiographic stimuli explains depressed individuals' uniquely dysphoric reaction to this type of stimulus.

Task Engagement

Group did not exert an overall effect on reported ability to execute task instructions, $F(2, 63) = 2.46, p > .05$. It is interesting to note that there was a Group \times Emotion interaction for this variable, $F(4, 126) = 2.67, p < .05$. Currently depressed participants' self-reported ability to execute task instructions differed between emotion conditions, $F(2, 17) = 5.78, p = .01$, with task execution lower in the sad ($M = 5.20, SE = .47$) and neutral ($M = 5.47, SE = .38$) conditions than the happy conditions ($M = 5.91, SE = .36$). In contrast, emotion condition had no impact on healthy control participants' and recovered depressed individuals' self-reported ability to execute task instructions: control participants, $F(2, 23) = 3.28, p > .05$; formerly depressed participants, $F(2, 19) = 3.45, p > .05$. It is notable that currently depressed persons reported greater difficulties with task execution during the sad tasks, but it is also important to note that currently depressed individuals' ratings of task execution were uncorrelated with their sadness reports during the sad conditions (all $ps > .1$). Therefore, although currently depressed persons' low engagement with negative stimuli may provide a partial explanation for ECI, our data suggest that low engagement is unlikely to be a full explanation of this effect.

Discussion

Increasingly, theories and methods from affective science are being applied to depression to understand how this mood disorder alters emotional reactivity (e.g., Kring & Bachorowski, 1999). The primary objective of the present study was to provide a comprehensive test of the ECI hypothesis relative to competing views of

emotional reactivity in MDD (specifically, the positive attenuation and negative potentiation hypotheses). According to the ECI hypothesis, depressed individuals should fail to mount typical emotional responses when placed in differing emotional contexts. To examine the conditions under which ECI holds, the response domains to which it applies, and its association with clinical state, we measured the self-reported experiential, behavioral, and autonomic responses of currently depressed, formerly depressed, and healthy individuals to a battery of normative and idiographic stimuli that elicited happy, sad, and neutral states.

ECI and Positive Attenuation Hypotheses Were Partially Supported

Support for the ECI hypothesis was obtained in self-reports of sadness experience. Unlike healthy control participants, currently depressed individuals reported similar levels of sadness in response to stimuli that differed in emotional valence. This failure to exhibit valence-modulation is consistent with ECI and is at odds with the negative potentiation hypothesis. Another major group difference was observed in self-reports of happiness experience, where depressed individuals exhibited lower levels of happiness than did participants in the other groups throughout the stimulus battery. It is important to note that no group differences in reported happiness reactivity were observed: Currently depressed individuals reported happiness appropriately in response to changing stimulus valence. That depressed persons reported less happiness across all contexts irrespective of valence is, on the surface, consistent with the idea of emotional inflexibility (ECI) and with a general lack of hedonic tone (positive attenuation) in MDD. Nevertheless, it must also be emphasized that reported happiness data evinced no group differences in reactivity. Thus, it is apparent that these self-reported happiness data do not permit a clear choice between the positive attenuation and the ECI views of emotional reactivity in MDD; additional empirical tests will be required to disambiguate these views.

Emotional Reactivity Effects Are Mood-State Dependent

Inconsistent with our expectation that ECI would be traitlike, there was no evidence that ECI characterizes formerly depressed individuals. In fact, we found that formerly depressed individuals actually exhibited more pronounced valence modulation of zygomatic EMG than did participants in the other two groups. This hyper-hedonic effect stands in contrast to our expectation that formerly depressed persons would exhibit ECI. It should be noted, however, that prospective studies of currently depressed individuals have found that both high levels of zygomatic EMG (Greden, Price, Genero, Feinberg, & Levine, 1984) and high levels of observer-rated happiness behavior displayed in an amusing context (Rottenberg, Kasch, et al., 2002) predict subsequent recovery from depression. It is possible, therefore, that our sample contained many individuals with relatively robust hedonic functioning, given that the formerly depressed individuals who participated in this study were required to be virtually asymptomatic, an atypical status for individuals who have a history of depression (Keller et al., 1992). Thus, the hyper-hedonic behavior observed in our sample of formerly depressed individuals may have been an indication of the unusual resilience exhibited by these select individ-

uals and might not have been observed had we included individuals with residual depressive symptoms.

More generally, formerly depressed individuals exhibited few abnormalities and largely resembled healthy control participants in their patterns of emotional reactivity. This is consistent with some questionnaire studies suggesting that depression-related deficits in emotional reactivity normalize with recovery (Blanchard, Horan, & Brown, 2001). At the same time, our finding that abnormalities in emotional reactivity are mood state dependent appears to be at variance with prior laboratory studies of emotional reactivity in MDD, which have found abnormalities to be mood state independent (e.g., Dichter et al., 2004). We should note that our study differs from prior studies, in both the broad range of stimuli that were used and the care with which we screened formerly depressed persons to ensure that they were free of residual depressive symptoms. In our view, the use of asymptomatic individuals is critical to test whether emotional reactivity in MDD is trait-like, but we concede that this complicates comparisons with studies using more liberal definitions of recovery from depression.

ECI and Emotion Response Domain

The present study replicated our prior work in other samples of depressed individuals (Rottenberg, Kasch, et al., 2002) in obtaining ECI effects only in reported emotional experience reactivity. As we noted earlier, other investigators have obtained ECI effects in behavior and in physiology (e.g., Allen et al., 1999). There are at least three possible reasons why investigators have observed evidence of ECI in different domains of emotional reactivity across studies.

First, depression-related ECI effects might, in fact, be present across emotion reactivity domains, but the current experimental paradigm may only have had adequate power to detect these effects in the domain of self-reported experience. Although this explanation cannot be discounted completely, we should point out that the present experiment did detect group effects in behavior (as measured by EMG), as well as the expected valence effects in both EMG and autonomic physiology.

Second, these discrepancies might be due to a characteristic of depressed individuals that varies systematically across studies. For example, depressed samples often vary in their extent of comorbid anxiety, a factor known to influence psychophysiology. Moreover, depression severity also varies across samples. Notably, several studies that obtained ECI effects in behavior and physiology were conducted with severely depressed inpatient samples (e.g., Allen et al., 1999; Dawson et al., 1977; Greden et al., 1986), whereas the current study assessed a more moderately depressed outpatient sample. Both depression severity (Rottenberg, Kasch, et al., 2002) and the presence of the melancholic subtype (Allen et al., 1999) have been associated with ECI, and these factors warrant further systematic investigation.

Finally, it is possible that ECI in depression is indeed specific to the domain of subjective emotion experience. If so, MDD would take its place alongside other disorders that clinical researchers have shown to be characterized by domain-specific emotional deficits. Perhaps the strongest findings of domain-specificity have been obtained with schizophrenic individuals, who have been described as having relatively intact subjective experience and physiology but blunted emotional behavior (e.g., Kring & Neale,

1996). Certainly, additional studies will need to replicate the present domain-specific pattern before reaching this conclusion for MDD.

Type Effects: The Significance of Idiographic Stimuli

Our study was the first to study emotional reactivity to both normative and idiographic stimuli in carefully defined depressed and remitted individuals. In this context, it is significant that depressed individuals' reports of sadness and happiness were *more* sensitive than were reports of participants in the other groups to changes in the personal concern of the stimulus. Overall, depressed persons appeared to exhibit a dysphoric response to idiographic stimuli that was observed broadly across emotion conditions. Indeed, the self-system of depressed individuals has been described as a highly interconnected network of negative content (Beck, 1967; Beck et al., 1979), and negative self-referential effects have been demonstrated in a large number of cognitive tasks. For example, in adjective rating tasks, depressed participants have been found to endorse more negative and fewer positive adjectives as self-descriptive than do nonpsychiatric control participants (e.g., Derry & Kuiper, 1981). It stands to reason that if idiographic stimuli differentially activate depressed persons' negative self-schemas, these stimuli should produce a more severe dysphoric state in depressed persons (i.e., more reported sadness and less reported happiness) than would normative comparison stimuli. Idiographic stimuli are also presumably more likely than normative stimuli to activate mood-congruent information in autobiographical memory, a process postulated to be important in depression maintenance (Blaney, 1986; Bower, 1981). It is interesting to note that exploratory analyses in this sample indicated that those depressed individuals who had greater differential responses to idiographic stimuli also tended to have longer episodes of depression. That is, those depressed individuals who reported less happiness and more sadness to idiographic than to normative stimuli tended to have been depressed for longer periods of time: happiness, $r(17) = -.47, p < .06$; sadness, $r(17) = .42, p < .1$. Prospective designs are warranted to determine the etiological significance of heightened responding to idiographic stimuli in MDD.

Explanations for ECI in Depression

One of the goals of the current study was to address artifactual explanations of ECI. The present results suggest that ECI is unlikely to be an artifact of a particular experimental setting. Depressed persons' ECI of sadness reports was observed broadly, across two types of stimuli (normative, idiographic), presented through two different modes (internal, external), and in conditions propitious for the alternative potentiation hypothesis (i.e., sad, idiographic, and internal). Secondary analyses also suggested that two other methodological explanations were unlikely to provide full explanations of ECI: Depressed persons did not perceive sad stimuli as irrelevant to their personal concerns, nor was their ability to follow task instructions related to their sadness reports.

If ECI is not an experimental artifact, what gives rise to ECI? The broader theory of ECI—that severe mood states have functional significance in reducing motivated activity—is relatively silent concerning proximal mechanisms (Rottenberg & Gotlib,

2004). We have speculated that ECI may reflect impairments in the capacity to offset negative emotions (Rottenberg & Gross, 2003), which may operate by means of faulty emotion regulation strategies (e.g., rumination; Nolen-Hoeksema, 1991) and/or deficiencies in homeostatic mechanisms in MDD (e.g., vagal tone; Rottenberg, Wilhelm, Gross, & Gotlib, 2003). Clearly, developing accounts of responsible proximal mechanisms—including the neural substrates for ECI—will be a high priority for future work.

Limitations and Conclusions

Several limitations of this study should be noted. First, the sample size was relatively modest. Our small group sizes precluded a detailed examination of the effects of gender, medication, or comorbid anxiety disorders. Second, although the 12 experimental conditions afforded a broad set of stimulus conditions, only three emotional states (happy, sad, and neutral) were probed. Other emotional states (e.g., anger) should be investigated with the same level of care. Third, only one mental disorder (MDD) was evaluated. Although effects were specific to individuals with current MDD symptoms, a demonstration of diagnostic specificity would require a psychopathology control group. Finally, this study was cross-sectional and could not address hypotheses regarding the course of depression. Longitudinal designs that follow the same individuals (including those in the present sample) across changes in clinical status will be crucial in further addressing the etiological significance of ECI.

In closing, we would like to note several strengths of this study. Emotion was probed in carefully diagnosed participants with a comprehensive stimulus battery. In testing three competing views of emotional reactivity in MDD, we found that the ECI and the positive attenuation hypotheses received limited support, whereas the negative potentiation hypothesis received no support: We replicated findings indicating that depressed individuals exhibit ECI in their reports of sadness experience, and we provided the first evidence that individuals with MDD are differentially sensitive to idiographic stimuli. This study thus represents an important first step in delineating the precise ways in which MDD alters emotional reactivity. We hope other investigators will refine the ECI hypothesis and add to these findings, thereby improving the diagnosis, the treatment, and, ultimately, the prevention of MDD.

References

- Allen, N. B., Trinder, J., & Brennen, C. (1999). Affective startle modulation in clinical depression: Preliminary findings. *Biological Psychiatry*, *46*, 542–550.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row.
- Beck, A. T. (1983). Cognitive therapy of depression: New perspectives. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depression: Old controversies and new approaches* (pp. 265–290). New York: Raven Press.
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, *56*, 893–897.
- Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.
- Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100.
- Beckham, E. E., Leber, W. R., & Youll, L. K. (1995). The diagnostic classification of depression. In E. E. Beckham & W. R. Leber (Eds.), *Handbook of depression* (pp. 36–60). New York: Guilford Press.
- Berenbaum, H., & Oltmanns, T. F. (1992). Emotional experience and expression in schizophrenia and depression. *Journal of Abnormal Psychology*, *101*, 37–44.
- Blanchard, J. L., Horan, W. P., & Brown, S. A. (2001). Diagnostic differences in social anhedonia: A longitudinal study of schizophrenia and major depressive disorder. *Journal of Abnormal Psychology*, *110*, 363–371.
- Blaney, P. H. (1986). Affect and memory: A review. *Psychological Bulletin*, *99*, 229–246.
- Blatt, S. J., Quinlan, D. M., Chevron, E. S., McDonald, C., & Zuroff, D. (1982). Dependency and self-criticism: Psychological dimensions of depression. *Journal of Consulting and Clinical Psychology*, *50*, 113–124.
- Bower, G. H. (1981). Mood and memory. *American Psychologist*, *36*, 129–148.
- Brown, S. L., Schwartz, G. E., & Sweeney, D. R. (1978). Dissociation of self-reported and observed pleasure in depression. *Psychosomatic Medicine*, *40*, 536–548.
- Canli, T., Sivers, H., Thomason, M., Whitfield, S., Gabrieli, J. D. E., & Gotlib, I. H. (2004). Brain activation to emotional words in depressed versus healthy subjects. *NeuroReport*, *15*, 2585–2588.
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*, 316–336.
- Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, *103*, 103–116.
- Dawson, M. E., Schell, A. M., & Catania, J. J. (1977). Autonomic correlates of depression and clinical improvement following electroconvulsive shock therapy. *Psychophysiology*, *14*, 569–578.
- Deldin, P. J., Keller, J., Gergen, J. A., & Miller, G. A. (2001). Cognitive bias and emotion in neuropsychological models of depression. *Cognition & Emotion*, *15*, 787–802.
- Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*, *40*, 457–492.
- Derry, P. A., & Kuiper, N. A. (1981). Schematic processing and self-reference in clinical depression. *Journal of Abnormal Psychology*, *90*, 286–297.
- Dichter, G. S., Tomarken, A. J., Shelton, R. C., & Sutton, S. (2004). Early- and late-onset startle modulation in unipolar depression. *Psychophysiology*, *41*, 433–440.
- Dickens, C., McGowan, L., & Dale, S. (2003). Impact of depression on experimental pain perception: A systematic review of the literature with meta-analysis. *Psychosomatic Medicine*, *65*, 369–375.
- Ekman, P. (1992). An argument for basic emotions. *Cognition & Emotion*, *6*, 169–200.
- Endicott, J., Spitzer, R. L., Fleiss, J. L., & Cohen, J. (1976). The Global Assessment Scale: A procedure for measuring overall severity of psychiatric disturbance. *Archives of General Psychiatry*, *33*, 766–771.
- First, M. B., Gibbon, M., Spitzer, R. L., & Williams, J. B. W. (1995). *User's guide for the structured clinical interview for DSM-IV Axis I disorders* (SCID-I, Version 2.0, October 1995 Final Version). New York: Biometrics Research.
- Fridlund, A. J., & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. *Psychophysiology*, *23*, 567–583.
- Gehricke, J. G., & Shapiro, D. (2000). Reduced facial expression and social context in major depression: Discrepancies between facial muscle activity and self-reported emotion. *Psychiatry Research*, *95*, 157–167.
- Golin, S., Hartman, S. A., Klatt, E. N., Munz, K., & Wolfgang, G. L.

- (1977). Effects of self-esteem manipulation on arousal and reactions to sad models in depressed and nondepressed college students. *Journal of Abnormal Psychology*, *86*, 435–439.
- Gotlib, I. H. (1984). Depression and general psychopathology in university students. *Journal of Abnormal Psychology*, *93*, 19–30.
- Gotlib, I. H., Sivers, H., Canli, T., Kasch, K. L., & Gabrieli, J. D. E. (2001, November). Neural activation in depression in response to emotional stimuli. In I. H. Gotlib (Chair), *New directions in the neurobiology of affective disorders*. Symposium presented at the Annual Meeting of the Society for Research in Psychopathology, Madison, WI.
- Greden, J. F., Genero, N., Price, H. L., Feinberg, M., & Levine, S. (1986). Facial electromyography in depression. *Archives of General Psychiatry*, *43*, 269–274.
- Greden, J. F., Price, H. L., Genero, N., Feinberg, M., & Levine, S. (1984). Facial EMG activity levels predict treatment outcome in depression. *Psychiatry Research*, *13*, 345–352.
- Healy, D. (1993). Dysphoria. In C. G. Costello (Ed.), *Symptoms of depression* (pp. 23–42). New York: Wiley.
- Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, *100*, 535–545.
- Henriques, J. B., & Davidson, R. J. (2000). Decreased responsiveness to reward in depression. *Cognition and Emotion*, *14*, 711–724.
- Iacono, W. G., Lykken, D. T., Haroian, K. P., Peloquin, L. H., Valentine, R. H., & Tuason, V. B. (1984). Electrodermal activity in euthymic patients with affective disorders: One-year retest stability and the effects of stimulus intensity and significance. *Journal of Abnormal Psychology*, *93*, 304–311.
- Judd, L. L., Paulus, M. P., Wells, K. B., & Rapaport, M. H. (1996). Socioeconomic burden of subsyndromal depressive symptoms and major depression in a sample of the general population. *American Journal of Psychiatry*, *153*, 1411–1417.
- Kasch, K. L., Rottenberg, J., Arnow, B. A., & Gotlib, I. H. (2002). Behavioral activation and inhibition systems and the severity and course of depression. *Journal of Abnormal Psychology*, *111*, 589–597.
- Keller, M. B., Lavori, P. W., Mueller, T. I., Endicott, J., Coryell, W., Hirschfeld, R. M., & Shea, T. (1992). Time to recovery, chronicity, and levels of psychopathology in major depression: A 5-year prospective follow-up of 431 subjects. *Archives of General Psychiatry*, *49*, 809–816.
- Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition & Emotion*, *13*, 467–480.
- Kessler, R. C. (2002). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (pp. 23–42). New York: Guilford Press.
- Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological Review*, *82*, 1–25.
- Kring, A. M., & Bachorowski, J. (1999). Emotions and psychopathology. *Cognition & Emotion*, *13*, 575–599.
- Kring, A. M., & Neale, J. M. (1996). Do schizophrenic patients show a disjunctive relationship among expressive, experiential, and psychophysiological components of emotion? *Journal of Abnormal Psychology*, *105*, 249–257.
- Lader, M. H., & Wing, L. W. (1969). Physiological measures in agitated and retarded depressed patients. *Journal of Psychiatry Research*, *7*, 89–100.
- Lang, P. J. (1978). Anxiety: Toward a psychophysiological definition. In H. S. Akiskal & W. L. Webb (Eds.), *Psychiatric diagnosis: Exploration of biological criteria* (pp. 365–389). New York: Spectrum.
- Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, *16*, 495–512.
- Larsen, J. T., Norris, C. J., & Cacioppo, J. T. (2003). Effects of positive and negative affect on electromyographic activity over *zygomaticus major* and *corrugator supercilli*. *Psychophysiology*, *40*, 776–785.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lewinsohn, P. M., Lobitz, W. C., & Wilson, S. (1973). “Sensitivity” of depressed individuals to aversive stimuli. *Journal of Abnormal Psychology*, *81*, 259–263.
- Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry*, *57*, 14–20.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, *97*, 569–582.
- Rosenberg, E. L. (1998). Levels of analysis and the organization of affect. *Review of General Psychology*, *2*, 247–270.
- Rottenberg, J., & Gotlib, I. H. (2004). Socioemotional functioning in depression. In M. Power (Ed.), *Mood disorders: A handbook of science and practice* (pp. 61–77). New York: Wiley.
- Rottenberg, J., & Gross, J. J. (2003). When emotion goes wrong: Realizing the promise of affective science. *Clinical Psychology: Science and Practice*, *10*, 227–232.
- Rottenberg, J., Gross, J. J., Wilhelm, F. H., Najmi, S., & Gotlib, I. H. (2002). Crying threshold and intensity in major depressive disorder. *Journal of Abnormal Psychology*, *111*, 302–312.
- Rottenberg, J., Kasch, K. L., Gross, J. J., & Gotlib, I. H. (2002). Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. *Emotion*, *2*, 135–146.
- Rottenberg, J., Ray, R. D., & Gross, J. J. (in press). Emotion elicitation using films. In J. A. Coan & J. J. B. Allen (Eds.), *The handbook of emotion elicitation and assessment*. London: Oxford University Press.
- Rottenberg, J., Wilhelm, F. H., Gross, J. J., & Gotlib, I. H. (2003). Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals. *Psychophysiology*, *40*, 1–6.
- Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., & Klerman, G. L. (1976, April 30). Facial muscle patterning to affective imagery in depressed and nondepressed subjects. *Science*, *192*, 489–491.
- Sigmon, S. T., & Nelson-Gray, R. O. (1992). Sensitivity to aversive events in depression: Antecedent, concomitant, or consequent? *Journal of Psychopathology and Behavioral Assessment*, *14*, 225–246.
- Sloan, D. M., Strauss, M. E., Quirk, S. W., & Sajatovic, M. (1997). Subjective and expressive emotional responses in depression. *Journal of Affective Disorders*, *46*, 135–141.
- Sloan, D. M., Strauss, M. E., & Wisner, K. L. (2001). Diminished response to pleasant stimuli by depressed women. *Journal of Abnormal Psychology*, *110*, 488–493.
- Thomas, K. M., Drevets, W. C., Dahl, R. E., Ryan, N. D., Birmaher, B., Eccard, C. H., et al. (2001). Amygdala response to fearful faces in anxious and depressed children. *Archives of General Psychiatry*, *58*, 1057–1063.
- Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology & Sociobiology*, *11*, 375–424.
- Watson, D. (2000). *Mood and temperament*. New York: Guilford Press.
- Wexler, B. E., Levenson, L., Warrenburg, S., & Price, L. H. (1994). Decreased perceptual sensitivity to emotion-evoking stimuli in depression. *Psychiatry Research*, *51*, 127–138.
- Wilhelm, F. H., Grossman, P., & Roth, W. T. (1999). Analysis of cardiovascular regulation. *Biomedical Science Instrumentation*, *35*, 135–140.

Received September 2, 2003

Revision received June 29, 2005

Accepted July 11, 2005 ■